single most important method of slowing the spread of HIV within populations, with mathematical modelling indicating that eliminating high infectivity in early infection has more effect than at any other disease stage.[5]

Thus, the diagnosis of PHI in at-risk individuals has considerable advantages in both individual and public heath terms. These two cases demonstrate how easy it can be to disregard such patients as having factitious HIV infection and are a gentle reminder that a negative antibody test does not necessarily exclude PHI. Healthcare professionals must continue to be alert to the less common clinical manifestations of PHI, be aware of the particular assays used in their own laboratory, and because no combination of symptoms is 100% sensitive or specific, diagnostic procedure must be broad and inclusive.[6]

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References

1 **Churchill DR**, De Cock KM, Miller RF. Feigned HIV infection/AIDS: malingering and Munchausen's syndrome. *Genitourin Med* 1994;**70**:314–16.
2 **Castilla J**, Sobrino P, De La Fuente L, *et al*. Late diagnosis of HIV infection in the era of highly active antiretroviral therapy: consequences for AIDS incidence. *AIDS*, 2002;16, 1945–51.
3 **Vernazza PL**, Eron JJ, Fiscus SA, *et al*. Sexual transmission of HIV: infectiousness and prevention. *AIDS* 1999;**13**:155–66.
4 **Leynaert B**, Downs AM, de Vincenzi I. Heterosexual transmission of human immunodeficiency virus: variability of infectivity throughout the course of infection. European Study Group on Heterosexual Transmission of HIV. *Am J Epidemiol* 1998;**148**:88–96.
5 **Koopman JS**, Jacquez JA, Welch GW, *et al*. Role of the Primary Infection in epidemics of HIV infection in gay cohorts. *J Acquir Immune Defic Syndr* 1994;**7**:1169–8.
6 **Kahn JO**, Walker BD. Acute human immunodeficiency virus type 1 infection. *N Engl J Med* 1998;**339**:33–9.

Complementary therapy and genital warts

Complementary therapy (CT) is now the second biggest growth industry in Europe (after IT). Up to 20% of the UK population visit a complementary therapist each year and as much as £5 billion is spent annually on such therapies.[1] In the United States this figure is $30 billion. The National Institutes of Health in the United States are keen to fund good scientific studies showing efficacy of CT, in order to ''disseminate authoritative information to the public and professionals''.[2] Objective data gathering is all the more important as a large majority of physicians view CT very negatively.[3]

Five years ago we were approached by a group of Reiki therapists to undertake a study showing the efficacy of Reiki healing on STIs. Reiki healing (RH) is a hands-on healing method that may be undertaken as distance healing.[4] There is a precedent for CT therapies being used in the form of yoga for patients with infection—in particular a well designed randomised trial showing efficacy in tuberculosis.[5] In view of this we undertook a study of the effect of RH at a distance on genital warts. The study had local ethics committee approval.

Patients with anogenital warts who were awaiting surgical treatment initially had their wart size and number assessed by a nurse using standard techniques.[6] Waiting time from this point to surgical removal of the warts averaged 6 weeks (plus or minus 1 week). Another nurse, who was blind to the initial wart visualisation, photographed the back of patient's head and then allocated each patient to a treatment (RH) or no treatment group according to a random code .Twelve Reiki healers were then each sent the photographs and undertook RH on them at a distance on a daily basis for about 10 minutes. Thus, half the patients received RH and the other half did not. Just before surgical removal of the warts the size and number of the warts was again assessed by the original nurse.

Considering a difference between a 35% reduction in wart volume for the Reiki treated group and a 10% reduction for the placebo (90% power 0.05) it was considered that 130 patients would be needed (65 in each arm); in fact, only 27 patients were enrolled into the study. Ten were lost to follow up .Of the 17 who completed the study nine received RH and eight did not. Two patients who received RH and one who did not totally cleared their warts. Seven who received RH and two who did not had an increase in wart mass/number. No patient who received RH and five who did not showed some degree of decrease in wart mass/number. These rates of regression are similar to those described in the placebo arms of recent double blind trials.[7 8]

Although this is a small study, we believe it was well designed but we failed to enrol large enough numbers. We also think it failed to show any efficacy for RH. Undertaking well designed trials of CT in the STI arena is important—not least because a majority of patients attending STI clinics may already be using them, and open discussion about them can help patients to make informed decisions as well as avoid drug interactions.[9]

In terms of common skin warts, efficacy of Reiki healing has not been shown to be effective.[10]

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References

1 **What Medicine**. CAM on the up as more people look for an alternative (www.whatmedicine.co.uk/articlesCompMed.htm).
2 **Anonymous**. About the National Center for Complementary and Alternative Medicine. (/nccam.nih.gov/aboutnccam/index.htm).
3 **Risberg T**, Kolsta A, Bremmes Y, Knowledge of and attitudes toward complementary and alternative therapies ; a national multicentre study of oncology professionals in Norway, *et al*. *Eur J Cancer* 2004;**40**:529–35.
4 **Mansour AA**, Beuche M, Laing G, *et al*. A study to test the effectiveness of placebo Reiki standardised procedures developed for a planned Reiki efficacy study. *J Altern Complement Med* 1999;**5**:153–64.
5 **Visweswaraiah NK**, Telles S. Randomised trial of yoga as a complementary therapy for pulmonary tuberculosis. *Respirology* 2004;**9**:96–101.
6 **Lacey CJN**, Goodall R, Tennvall GT, *et al*. Randomised controlled trial and economic evaluation of podophyllotoxin solution, podophyllotoxin cream, and podophyllin in the treatment of genital warts. *Sex Transm Infect* 2003;**79**:270–5.
7 **Tyring S**, Edwards L, Cherry K, *et al*. Safety and efficacy of 0.5% podofilox gel in the treatment of anogenital warts. *Arch Dermatol* 1998;**134**:33–8.
8 **Edwards L**, Ferenczy A, Eron L, *et al*. Self-administered topical 5% imiquimod cream for external anogenital warts. *Arch Dermatol* 1998;**134**:25–30.
9 **Trutnovsky G**, Law C, Simpson JM, *et al*. Use of complementary therapies in a sexual health clinic setting. *Int J STD AIDS* 2001;**12**:307–9.
10 **Harkness EF**, Abbot NC, Ernst E. A randomised trial of distant healing for skin warts. *Am J Med* 2000;**108**:507–8.

Chlamydia trachomatis PCR positivity and inflammatory changes on cervical cytology

The presence of genital infection does not increase the likelihood of an inadequate Papanicolaou (Pap) test.[1] Conversely, testing for *Chlamydia trachomatis* at the time of routine cytological screening presents an opportunity to detect asymptomatic genital tract infection.[2] The PreservCyt fixative fluid (Cytyc Corporation, Boxborough. MA, USA) used for the ThinPrep Pap test (Cytyc Corporation) can be used for detection by the polymerase chain reaction (PCR) of *C trachomatis*.[3 4] This presents an opportunity to study the correlation between the chlamydia result and the Pap test finding.

We retrospectively reviewed all routine requests for chlamydia PCR on ThinPrep samples sent to our laboratory over a year. Data were collected on the woman's age, chlamydia PCR result, result of genital tract cultures if performed on the same date, and Pap test result. Data on the Pap test result included presence or absence of an epithelial cell abnormality either high grade (HGEA) or low grade (LGEA), whether the Pap was inflammatory and the presence or absence of recognisable pathogens. Cervical specimens collected in PreservCyt transport medium were processed for *C trachomatis* using the automated Cobas Amplicor (Roche Diagnostic Systems) and the method by Bianchi *et al*.[3]

Over the study period, 733 samples were received, of which 23 (3.1%) had *C trachomatis* DNA detected by PCR. Comparison of the women with chlamydia infection, with those without chlamydia infection is shown in table 1. There was no statistical difference in the presence of high or low grade epithelial abnormalities, recognition of other pathogens, or age of the women; however, 26% of women with chlamydia had an inflammatory Pap test compared to 9% of women without chlamydia ($p<0.01$).

Table 1 Comparison of women with and without chlamydia infection

	Positive *C trachomatis* PCR	Negative *C trachomatis* PCR	p Value
Median age	24 (range 19–40)	28 (range 15–68)	0.182
LGEA/HGEA	5 (22%)	106 (15%)	0.37
Other pathogens	1 (17%)	12 (18%)	NS
Inflammation on Pap test	6 (26%)	65 (9%)	<0.01

LGEA, low grade epithelial abnormalities; HGEA, high grade epithelial abnormalities.

The association of inflammation on Pap testing and chlamydial infection has been previously examined with variable methodologies and findings.[5] We utilised the same sample (ThinPrep) for determining both the presence of inflammatory changes on Pap test and chlamydia infection and found a positive association between the two despite a low prevalence population. Our study confirms the feasibility of performing chlamydia PCR from liquid based cytology samples in a routine diagnostic setting. Testing for chlamydia should be considered in women with inflammatory Pap tests for which there is no other explanation.

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References

1 **Edwards S**, Sonnex C. Influence of genital infection on cervical cytology. *Sex Transm Infect* 1998;**74**:271–3.
2 **Hopwood J**, Mallinson H, Hodgson E, *et al*. Liquid based cytology: examination of its potential in a chlamydia screening programme. *Sex Transm Infect* 2004;**80**:371–3.
3 **Bianchi A**, Moret F, Desrues JM, *et al*. PreservCyt transport medium used for the ThinPrep Pap Test is a suitable medium for detection of Chlamydia trachomatis by the Cobas Amplicor CT/NG test: results of a preliminary study and future implications. *J Clin Microbiol* 2002;**40**:1749–54.
4 **Koumans EH**, Black CM, Markowitz LE, *et al*. Comparison of methods for detection of Chlamydia trachomatis and Neisseria gonorrhoeae using commercially available nucleic acid amplification tests and a liquid Pap smear medium. *J Clin Microbiol* 2003;**41**:1507–11.
5 **Paler RJ**, Simpson DR, Kaye AM, *et al*. The relationship of inflammation in the papanicolaou smear to Chlamydia trachomatis infection in a high-risk population. *Contraception* 2000;**61**:231–4.

Cardiovascular syphilis in HIV infection: a case-control study at the Institute of Sexually Transmitted Diseases, Chennai, India

It is known that HIV co-infection with syphilis may accelerate the onset of gummata and neurosyphilis and increase their severity. However, this has only been reported for cardiovascular syphilis in two previous cases.[1 2]

This case-control study deals with a total of 14 HIV seropositive and 100 HIV 1 and 2 seronegative individuals with syphilis, who were seen in our clinic between June 2000 and May 2001. Of the 14 HIV seropositive individuals, 12 were reactive for VDRL (venereal diseases reference laboratory) and TPHA (*Treponema pallidum* haemagglutination assay) and two had primary syphilis confirmed by dark field examination for *T pallidum*. Of the 100 HIV seronegative individuals, 85 had reactive VDRL and TPHA and 15 had primary syphilis confirmed by dark field examination. The prevalence of cardiovascular syphilis in the HIV seropositive and seronegative groups was 14.3% and 2%, respectively (OR 8.2; 95% CI 1.1 to 61.5).

Two HIV seropositive individuals with cardiovascular syphilis had aortic root dilatation while the two HIV seronegative individuals had aortic aneurysm. The HIV seropositive individuals were asymptomatic with regard to cardiac status but one HIV seronegative individual had chest pain and the other was asymptomatic. None in the HIV seronegative group had aortic root dilatation ($p<0.01$). There was a theoretical possibility that aortic root dilatation could be a manifestation of HIV or opportunistic infections involving the heart. A parallel study done on cardiovascular involvement in HIV seropositive individuals from the same institute during the same time interval had revealed that none of the 61 non-syphilitic HIV seropositive individuals had aortic root dilatation, compared with 2 out of 14 with syphilis ($p<0.01$; paper in preparation).

The mean duration of diagnosing cardiovascular syphilis from the time of acquiring syphilis was 40 months (27 and 53) in the HIV seropositive group and 102 months (84 and 120) in the HIV seronegative group. The mean age of the HIV seropositive individuals who had cardiovascular syphilis was 31.5 years (29 and 34) and that of HIV seronegative individuals was 45.5 years (44 and 47).

The shorter duration for diagnosing cardio vascular syphilis from the time of acquiring syphilis for the HIV seropositive group (40 months) compared with the HIV seronegative group (102 months) ($p<0.003$) could be explained by the fact that HIV hastens the progression to late syphilis,[3] which might be due to an alteration to the immune system. It could also be possible that HIV infected individuals seek medical attention because of opportunistic infections, which might have led to the earlier diagnosis of cardiac lesions because the two individuals with aortic root dilatation were asymptomatic with regard to cardiac status. The difference in the clinical manifestation of cardiovascular syphilis between these two groups could not be explained at this point of time.

Contributions

MM designed the study, collected the data, interpreted the results, and analysed the results and statistics; SKG contributed to collecting data, interpretation of results and laboratory collaboration.

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References

1 **Chetty R**, Batitag S, Nair R. Large artery vasculopathy in HIV-positive patients: another vasculitic enigma. *Hum Pathol* 2000;**31**:374–9.
2 **Olmos JM**, Fernandez-Ayala M, Gutierrez JA, *et al*. Superior vena cava syndrome secondary to syphilitic aneurysm of the ascending aorta in a human immunodeficiency virus-infected patient. *Clin Infect Dis* 1998;**27**:1331–2.
3 **Gregory N**, Sanchez M, Buchness MR. The spectrum of syphilis in patients with human immunodeficiency virus infection. *J Am Acad Dermatol* 1990;**22**:1061–7.

Antiretroviral therapy—alternative uses

Recently, while speaking to a patient from Nigeria I was very concerned to discover that she had been taking combivir for breast enhancement. On closer questioning it appears that she had accessed this drug, passed to her in individual sachets with no information insert etc, via a friend. Her friend, knowing that my patient wished for larger breasts, had passed her the combivir to use on an as required basis for breast enhancement. My patient claims that the drug did work to enlarge her breasts.

The drugs were apparently prescribed by a doctor in Nigeria at the cost of about US$250 for six sachets and the pharmacist dispensing them had been asking why the girls were taking them. Apparently the sachets did not come with any leaflets or drug information inserts.

My patient and her friends appeared to be totally unaware of the fact that the combivir was for use in HIV therapy and were unaware of any potential side effects from the drug. It was only when my patient was surfing the web that she found out about the licensed use for combivir.

My patient, sadly, acquired HIV from a blood transfusion in Africa and on primary resistance testing showed very broad nucleoside reverse transcriptase inhibitor resistance and apparent full sensitivity to protease inhibitors and non-nucleoside reverse transcriptase inhibitors (NNRTIs). Resistance to